

Supporting Information

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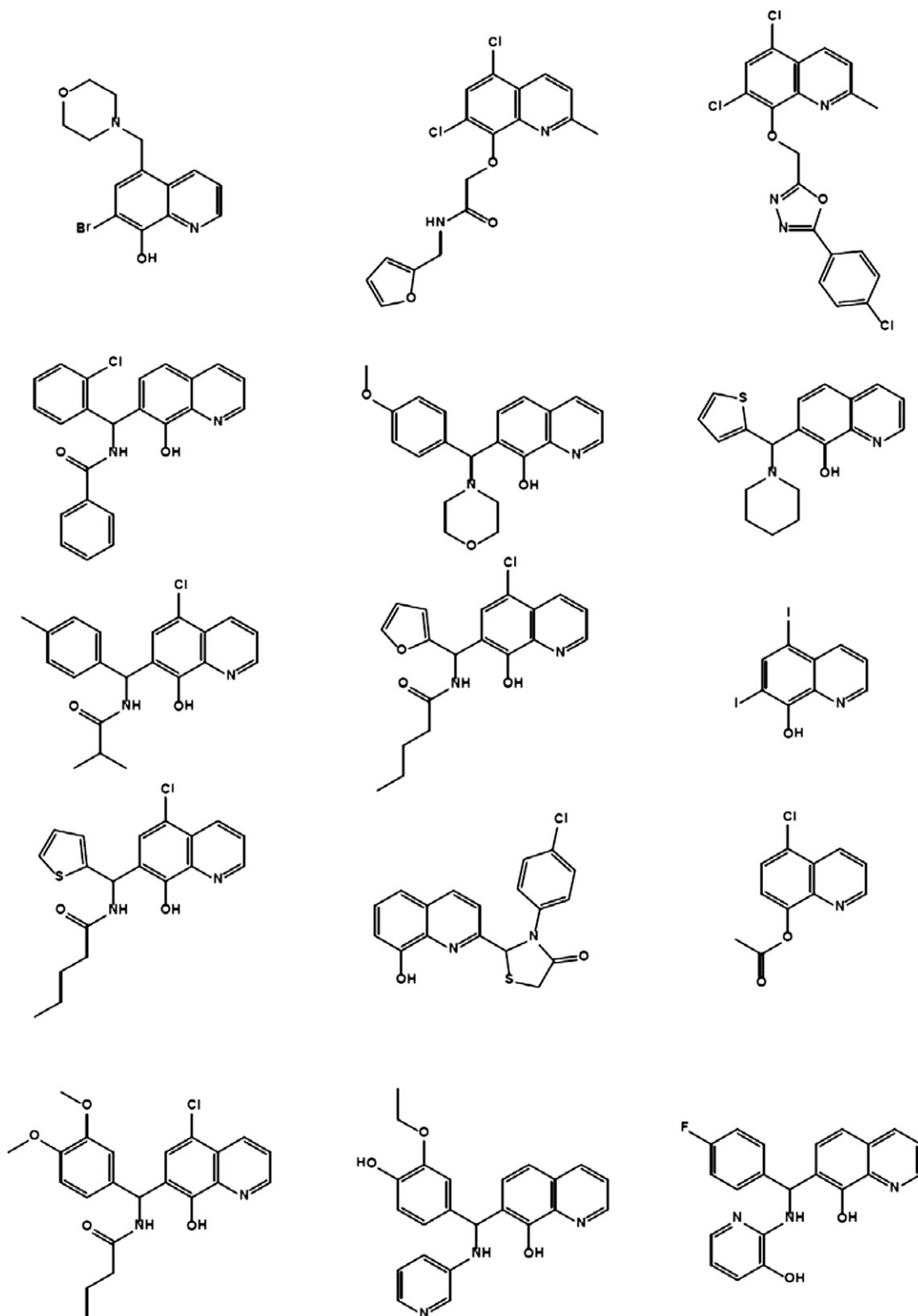


Fig. S1. Structures of 8-hydroxyquinoline (8-OHQ) hits. Fifteen of 30 hits from the initial β -amyloid ($A\beta$) small-molecule screen were 8-OHQs. All compounds had the core 8-OHQ with various, diverse substituents, largely attached through the hydroxyl-containing ring.

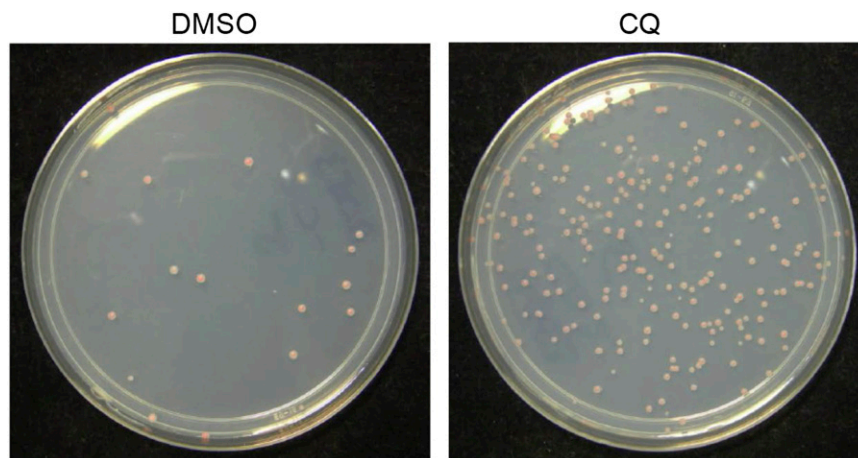


Fig. 52. Clioquinol (CQ) increases cell number. To ensure that the increase in OD_{600} was due to a rescue of toxicity and not due to an artifact of increased cell size, cells were plated to single colony-forming units after CQ treatment. Indeed, there was an increase in the number of viable cells after treating $A\beta$ -expressing cells with a protective dose of CQ.

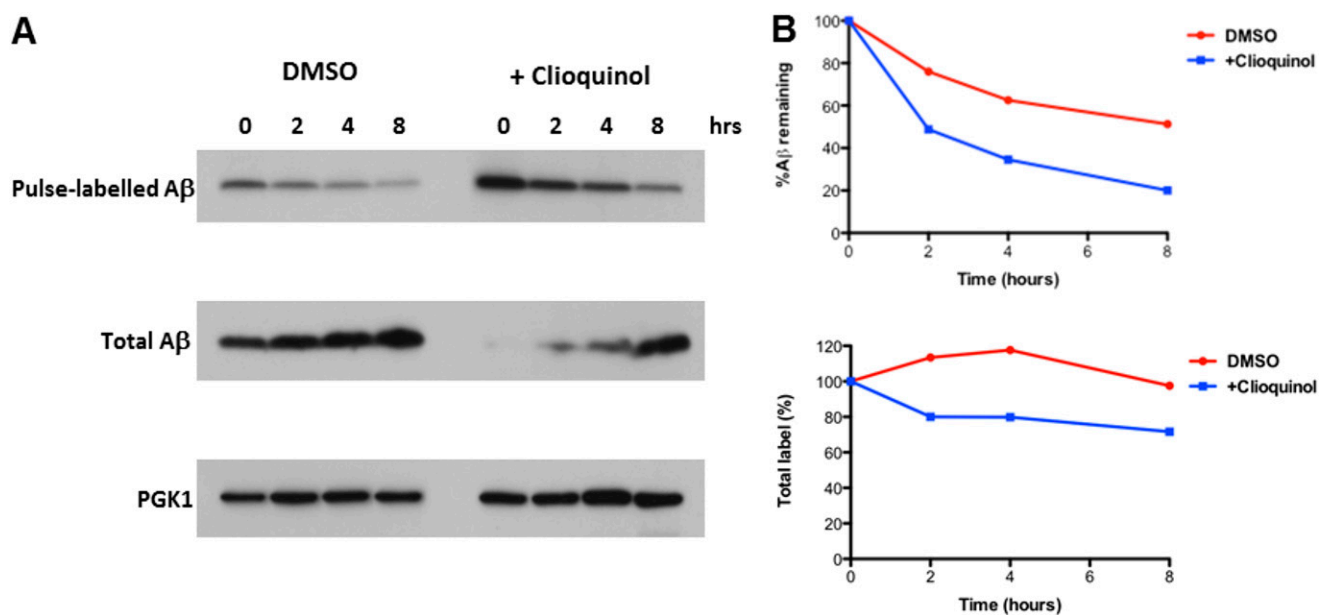


Fig. 53. ^{35}S pulse labeling of $A\beta$. (A) $A\beta$ analysis of pulse-labeling experiment. The top panel shows ^{35}S -labeled, immunoprecipitated $A\beta$ over time. The middle shows total $A\beta$ by immunoblot with the 6E10 antibody. The bottom panel shows levels of control protein, Pgk1. (B) Quantification of $A\beta$ and total ^{35}S labeling during degradation time course. The top panel is exact data shown in Fig. 6. The bottom panel shows total label as quantified from an entire lane of protein without immunoprecipitation.

Table S2. Gene deletions and toxic compound tested for rescue by CQ

Yeast gene deletion/drug	Gene name	Mechanism	Significant rescue by CQ?
Systematic yeast name			
YEL046C	GLY1		No
YGL070C	RPB9		No
YCR028C	FEN2		No
YDR463W	BAP1		No
YGR167W	CLC1		No
Drug			
Trifluoperazine		H ⁺ ATPase, K ⁺ regulation	No
<i>t</i> -Butyl hydrogen peroxide		Oxidant	No
Rapamycin		TOR inhibition	Mild
4-Nitroquinoline-1-oxide		DNA damage	No
Cycloheximide		Translation inhibitor	No
Gliotoxin		Thioredoxin redox	No
Hydroxyurea		dNTP depletion	No